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A child with upper airway infection in stridor: airway management

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ABSTRACT

Airway management of a two year old child with upper airway infection in stridor is presented to highlight the importance of understanding the dynamics of airway obstruction.

INTRODUCTION

Stridor is a high-pitched sound heard during respiration. It is produced due to turbulence in airflow, created by a partially obstructed airway. It is a sign of respiratory obstruction and all efforts must be directed to establish the cause. In children, stridor of an acute onset is often associated with upper airway infection or an inhaled foreign body [1]. Airway oedema occurring subsequent to instrumentation of the airway, tracheal intubation, trauma to upper airway, as from toxic ingestions; drug side effect or allergic reaction may also lead to stridor. Other causes of stridor are subglottic stenosis, subglottic haemangioma, vocal cord paralysis, laryngomalacia, laryngeal web, laryngeal papilloma or neoplasm [2].

CASE REPORT

A two-year-old boy was brought to paediatric emergency with history of sudden onset of noisy breathing while asleep and excessive crying since one hour. He had high grade fever, cough, change of voice and inability to accept feed since one day. There was no history suggesting foreign body aspiration. He was a known case of acyanotic heart disease and had past history of recurrent chest infections but was never hospitalized before. On examination he was febrile (102°F), irritable, had inspiratory stridor, flaring of ala nasi, supraclavicular and intercostal retraction. His heart rate was 130/minute and respiratory rate 54/minute. The chest had bilateral wheeze, coarse crepts and a systolic murmur was audible. Abdomen was soft, there was no organomegaly. All blood investigations were unremarkable except for an increase in total leucocyte count. X-rays of chest and neck (anteroposterior and lateral views) were unremarkable. On ENT examination the laryngeal mucosa was congested; the epiglottis was overhanging and congested. A provisional diagnosis of acute laryngotracheobronchitis (croup) or acute epiglottitis was made. Intensive medical therapy with humidified oxygen, nebulized adrenaline and salbutamol was started with the child in semi upright position. Injections ceftriaxone, dexamethasone, ranitidine and terbutaline were given. He showed no improvement and was shifted to the intensive care unit.

He was in respiratory distress, his heart rate was 170/minute, respiratory rate 62/minute, and SpO2 81% on room air and 93% on oxygen. With possibility of acute epiglottitis as one of the probable causes of respiratory distress, we decided to secure the airway immediately. Equipment for emergency airway management, endotracheal tubes of size smaller than expected for age, stylet, bougie, jet ventilator, and emergency tracheostomy was kept ready. After obtaining a written informed consent from the parents, anaesthesia was induced using halothane in oxygen with the child in semi upright position. On achieving sufficient depth of anaesthesia, he was reclined and laryngoscopy was done. The oropharynx and epiglottis appeared congested and oedematous. The trachea was intubated with orotracheal tube (uncuffed 3.5mm ID, Portex tube) with help of a stylet allowing a minimal acceptable air leak. After confirming bilateral equal air entry, the child was put on a ventilator and mechanically ventilated. He was given supportive care and medications as

started initially.

He was weaned off the ventilator and extubated on the second day, as peritubal air leak had increased suggesting subsiding of airway edema. Since, his breathing was laboured and stridor persisted, we re-intubated him. This time a bigger ETT (4mm ID) could be inserted allowing acceptable air leak. By the fourth day, besides a significant increase in peritubal air leak, the child was afebrile and had minimal clear tracheal secretions. His chest was clear on auscultation. Following extubation, he was breathing comfortably; there was no stridor or any sign of respiratory distress. The next day he was transferred to ward.

DISCUSSION

Upper airway infections in infants and small children can lead to airway obstruction due to narrowing of the airway produced by inflammation. Croup is preceded by upper respiratory tract infection and is the commonest cause of upper respiratory tract obstruction in children [3]. It is primarily of viral origin. It has male predominance and is mostly seen in the age group of 3 months to 3 years, with peak occurrence in second year of life [4]. The course of illness is usually mild & self-limiting. However, potentially life threatening airway obstruction may occur due to subglottic oedema, tracheal and bronchial inflammation and increased mucosal secretions [5]. Dexamethasone is the keystone of treatment and nebulization with epinephrine is added in severe croup [4]. The beneficial effects of mist have not been proven; there is insufficient evidence to support the use of heliox (helium-oxygen) in improving gas exchange [6-8]. Most of the cases can be managed conservatively; endotracheal intubation is required in only 1% of cases [9].

It may at times be very difficult to differentiate croup from acute epiglottitis, which is a life threatening emergency with a case fatality rate of 3 to 4 % [3, 10]. High grade fever, toxic appearance, rapidly progressive disease and drooling may suggest a bacterial infection; especially epiglottitis and bacterial tracheitis [4].

Whenever a diagnosis of acute epiglottitis is contemplated, the airway must be secured, as complete airway obstruction is a distinct possibility. It is advisable to secure the airway in an operation theatre or intensive care unit by an experienced anaesthesiologist, who is prepared for a difficult airway. An otolaryngologist must be available, should a surgical airway be required. The airway should not be manipulated until the plane of anaesthesia is deep. If the patient's airway worsens on reclining, induction of anaesthesia should take place in sitting position. It may be prudent to use inhalation techniques of induction of anaesthesia without intravenous induction agents or muscle relaxants to prevent loss of spontaneous ventilation. Intubation must be smooth and atraumatic to prevent aggravation of mucosal swelling and further worsening of airway obstruction. Once the airway is secured, intensive medical therapy with third generation cephalosporin is recommended [5]. The child must be kept sedated to avoid accidental extubation and permit ventilation.

Prompted by an increase in peritubal air leak as a sign of decreasing airway oedema, we prematurely extubated the trachea and had to re-intubate. Repeated intubations can cause tracheal trauma, increase mucosal swelling and cause further worsening of airway obstruction. Abatement of fever, decreased tracheal secretions, change in type of secretions to thin and watery apart from an audible peritubal air leak are other useful indicators of response to treatment, which need to be considered prior to tracheal extubation [11].

CONCLUSION

When aetiology of stridor in a child is upper airway infection, acute epiglottitis must remain foremost in the differential diagnosis. At the slightest suspicion, treatment should proceed as for acute epiglottitis, owing to fulminant nature of this disease with rapid progression to complete airway obstruction. Timely securing an artificial airway and tracheal extubation are both crucial steps in management and should be done with caution.

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